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Models of Schizotypy: The Importance of Conceptual Clarity

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The observation of psychosis-like traits that resemble symptoms of schizophrenia and bipolar disorder, both among healthy relatives of psychotic patients and among the general population, can be traced to the early 20th century.[1,2] These traits have since been described within various models of illness and health (ie, normal/abnormal personality, abnormal psychotic continua), each giving rise to concepts such as "schizotypy," "psychoticism," and "psychosis-proneness" that are not necessarily interchangeable, although their subtle distinctions are often overlooked. Historically, there have been 3 major models of schizophrenia-/psychosis-proneness, one of which is referred to as "taxonic" or "*quasi*-dimensional,"[3,4] and 2 models that can be regarded as "*fully* dimensional,"[5,6] as distinguished by the relationship that is proposed to exist between psychosis-proneness and the risk of clinical schizophrenia or other psychotic disorder. In this review, we outline the key assumptions of each model and its implications for research of psychosis in relation to mental illness and health and for the alternative models. We integrate historical concept development with current findings from various fields of research (eg, personality, neurobiology, and behavioral genetics) and highlight the remaining questions each model poses in relation to understanding the development of psychotic illness and the distribution of psychotic-like traits in the general population.

Key words: schizotypy/psychosis proneness/schizotypal personality/schizophrenia/schizotypy models

Introduction

Schizotypy is agreed to comprise a set of inherited traits reflected in personality organization,[3,4,6] which present as qualitatively similar to schizophrenia symptoms and correlate with schizophrenia liability. There is consensus that schizotypy is a multifaceted concept—though there remains a lack of consensus on its core dimensions and the relative import of each. For example, the consequences for schizophrenia liability of presenting with high values in one but not another schizotypal facet, or particular combinations of schizotypal traits, remain unclear.

The construct of schizotypy is increasingly accepted in the clinical sciences as an "*influential, comprehensive psychological construct in schizophrenia research*"[7] (p. S363) and a "*useful and unifying construct for understanding schizophrenia-spectrum psychopathology*"[8] (p. S366). Historically, schizotypy has been regarded as a set of personality traits distributed among (at least significant parts of) the general population, which may represent an "endophenotype" on the path to schizophrenia.[9,10] However, there remains considerable lack of conceptual clarity about schizotypy and its relevance in understanding the causes of psychotic disorder. We believe this partly reflects failure to acknowledge the historical development of the schizotypy construct, particularly, subtle differences among key theoretical models from which the construct emerged. This review highlights the key assumptions of various schizotypy models as they emerged over time, contributing to current concepts (and potential misunderstandings) about the use of the schizotypy construct. We review these different models and urge researchers in this field to consider these distinctions in theoretical foundations when reporting data concerning "schizotypy."

Meehlian Model

Historically, the notion of latent schizophrenia-like characteristics observable both in patients prior to their first florid episode and in patients' nonschizophrenic relatives can be traced at least back to the early 20th century.[1,2] Since then, a number of terms have been used to denote

the existence of psychotic-like experiences in nonpsychotic individuals; the term "schizotypy" (a contraction of "schizophrenic phenotype" introduced by Rado[3]) being the most commonly used. Rado's "schizotypy" was based on genetic liability and heavily built upon by Meehl.[4] Both authors proposed the existence of a discrete class of individuals (schizotypes) characterized by an integrative neural defect believed to be caused by a specific "schizo-gene" with a dominant Mendelian pattern of inheritance.[4] Although modern genetics has ruled out the idea that schizophrenia is a Mendelian disorder, nor likely caused by a single gene, Meehl proposed the importance of "polygenic potentiators" (see below) believed to influence a number of genetic factors that may interact with the proposed "schizogene" to determine the likelihood of transition to clinical schizophrenia.[11] One major misconception of Meehl's model has been in understanding the expected transition rates of schizotypes into schizophrenia: not all schizotypes were presumed to transition. Instead, according to the prevalence of schizophrenia, Meehl surmized that 10% of the population be regarded as schizotypes, but only 10% of these would decompensate into schizophrenia, while the other 90% would remain asymptomatic or show a subclinical expression of symptoms.

Furthermore, Meehl did not assume that schizotypy was (fully) inherited, rather that the phenotype emerged from gene-environment interactions. He specifically proposed that the aforementioned "schizogene" would lead to an integrative neural defect (schizotaxia), which *could* result in schizotypal personality organization (this not being synonymous with schizotypal personality *disorder*) dependent on individual environmental exposure and a range of genetically determined personality dimensions (independent of schizotaxia) referred to as "polygenic potentiators."[11] Thus, only schizotaxia (the neural integrative defect) was proposed to be inherited.[4] In other words, Meehl proposed that schizotaxia almost invariably leads to schizotypy and sometimes to schizophrenia—perhaps due to other genes, the learning environment etcetera. Importantly, the Meehlian model does not exclude influences of other genes than the "schizogene" on idiosyncratic expression of schizotypy, both as "potentiators" and "depotentiators (ie, influencing idiosyncratic schizotypal organization and altering the risk of decompensation, but only "*given the presence of the schizogene*"[11]; p. 39).

Although the single-gene aspect of Meehl's model is inconsistent with Genome Wide Association Studies (GWAS) of schizophrenia,[12–14] suggesting that the probability of a monogenetic cause of schizophrenia liability is highly unlikely, it has been asserted that the model is compatible with a polygenic basis of schizotaxia.[15] Others, however, have illustrated that an increasing number of involved alleles (with individually small effect-sizes) leads to the resulting quantitative trait becoming dimensional rather than taxonic.[16,17] Furthermore, a single risk-allele (or "schizogene") would need to have effects of an order of magnitude that makes it highly unlikely not to have been discovered by now. Importantly, in the genetic context, Meehl's model represents a taxonic one because it allows for phenotypic variation along a continuum of severity *within* schizotypy, but places the entire continuum within the realm of *illness* (associated with genetic predisposition). That is, all schizotypes are necessarily "schizotaxic," carrying of at least one copy of one or more risk alleles defining schizotaxia and, by extension, a schizotype. Thus, one either is a schizotype or not, but within the group of schizotypes, there is proposed gradation regarding symptom severity. Claridge[6] attempted to distinguish his *fully dimension* model of schizotypy (which allowed "schizotypy" to exist in both illness and health) by referring to Meehl's model as "quasi-dimensional." Thus, while Meehl's model allows schizophrenia risk to vary in severity on a dimension *within* a (clinical) taxon [schizotypy], Meehl did not believe that schizotypal personality extended outside of the taxon throughout the general population. Meehl's model, thus, represents a *quasi*-dimensional account because of the proposed clear demarcation between the healthy and schizotaxic brain: the abnormal brain state (schizotaxia) is taken as a reference point, and dimensions of the spectrum of schizotypal behaviors are construed as degrees of expression of "disorder," with the ultimate end-point being schizophrenia.[18] The most commonly used schizotypy scales developed within the framework of the Meehlian model are the Wisconsin Schizotypy Scales by the research team of Jean and Loren Chapman.

Eysenckian Model

In contrast to the Meehlian *disease-based* model, the "*fully dimensional*" view emerged from European school of temperament rooted in experimental psychology, particularly pioneered by Hans Eysenck.[5,19] Eysenck's theory saw psychotic illness as the extreme end of a continuous personality dimensions, couched within natural variation in brain functioning. At the time, Eysenck's proposal of an inextricable connection between normal and abnormal personality along with the assumption of biological causation dissected many issues within the debate between psychiatry and the antipsychiatry movement. Eysenck proposed that all major dimensions of personality were genetically based, interacted with the environment, and expressed themselves phenotypically via biological intermediaries (eg, hormones, neurotransmitters). It is important, therefore, to emphasize that—although Eysenck did not research individual genetic contributions—his theory (and by extension that of his former student, Claridge) is fully rooted in genetics.[20] Additionally, it is often misconstrued that Eysenck and his followers used statistical methods (ie, factor analysis) to reach theories (as is

the case, eg, regarding the Big Five personality model), while the opposite was true in actuality: Eysenck consistently maintained that personality research should always start with hypotheses and that experiments and statistical methods be used to test these hypotheses, not vice versa.[5,20] Thus, while modern personality models (eg, the Five Factor Model) are mainly data driven, Eysenck's approach was of a truly deductive (ie, theory driven) nature.

The Eysenckian model differs from the Meehlian not only in the assumption of *complete* dimensionality of schizophrenia liability but also in the assumption that there cannot be a single "pure dimension of schizotypy." Eysenck did not see room for its existence,[21] because it relied on the Kraepelin-Bleuler dichotomy of schizophrenia and bipolar disorder as qualitatively discrete entities. In other words, if schizotypy existed, it should be distinguishable from another trait one might call "cyclotypy." This notion was *prima facie* proposed by Kretschmer,[2] when he formulated his temperaments of schizothymia and cyclothymia. Kretschmer did not view these as discrete entities, however, rather as opposing expressions of the same trait; also assuming a continuum from normal to psychotic. On this notion, Eysenck convincingly argued that one "*cannot have a single dimension with 'psychosis' at both ends*"[22] (p. 767); instead, proposing the existence of 3 personality dimensions: ("Psychoticism", "Extraversion", and "Neuroticism"). According to this model, psychotic disorders are focal points of quantitative dimensions (ie, extreme values in Psychoticism combined with individual expressions of Extraversion/Neuroticism) and equivalent with clinical syndromes, though Eysenck largely eschewed psychiatric concepts.

Eysenck proposed that all clinical disorders were "*observed constellations […] of traits*"[19] (p. 28); in this view, "Psychoticism" was an aspect of general personality capturing the underlying dimensional liability for all psychotic disorders: keeping with the concept of *Einheitspsychose* (unitary psychosis). It is noteworthy that he cites Kraepelin himself as having potential doubts about the dichotomy of schizophrenia and bipolar disorder: "*it is becoming increasingly clear that we cannot distinguish satisfactorily between these two illnesses and this raises the suspicion that our formulation of the problem may be incorrect.*"[23] (cited in[22]; p. 758). Regarding the Eysenckian view of Psychoticism, however, there is an ongoing, heated debate (currently, tending to favor "the old Eysenck over the new"): It is commonly known—and often stressed by Lenzenweger[15,24]—that the current conceptualization of the Eysenck P-scale[25] bears little resemblance to traits understood as "schizotypal." Rather, modern Psychoticism captures cold heartedness, tough mindedness, low Agreeableness, impulsivity, and similar traits more related to psychopathy than psychosis; thus, reflecting the academic perception common to the times of schizophrenics being *inherently* prone to violence and delinquency. This is emphasized by Claridge, whose schizotypy model is built on the older conceptualization of Psychoticism. This older concept—although only ever (and very tentatively) published in out-of-print-books[5]—was far more closely related to psychosis than psychopathy. Furthermore, as the validity of separable functional psychoses is currently being brought into question, it is a very germane issue whether "schizotypal" traits are specific to schizophrenia or more generally relevant to psychosis. A related issue, and in fact a major weakness of Eysenck's account, is that it fails to make a clear distinction between traits and clinical states or offer any cogent explanation about how traits lead to illness. We have seen how Meehl's account approached this distinction and can now turn to Claridge's extension of a *fully* dimensional model of schizotypy.[6]

Claridge's Model

According to Claridge,[26] schizotypy denotes a range of enduring personality traits, reflected in cognitive style and perceptual experiences, arising from a combination of polygenetic and environmental determinants, which are normally distributed within the general population. An important distinction between the *fully* dimensional model proposed by Claridge[6,26] and Eysenck's *earlier* model is that the former proposes a boundary between health and illness along the schizotypy-schizophrenia continuum, where signs of discontinuity of function are used to denote abnormality (ie, disorder). For Claridge, schizotypal traits comprise dual properties insofar as they represent adaptive variation in personality but also comprise the potential for maladaptive functioning (ie, they are necessary but not sufficient for schizophrenia). Thus, high expressions of schizotypy are necessary for psychotic disorders, but it is an independent dimension (which Claridge suggestively called "health"[6]) that marks the risk of transition into illness. As such, Claridge's *fully* dimensional model of schizotypy[6,26] takes normal variation in personality as the starting point of the schizotypal spectrum, and this is also reflected in the scale composition of the associated Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE[27]). Claridge's model of schizotypy draws parallels between psychiatric illness and somatic disorders, using the example of hypertension as a template (ie, sustained high blood pressure brings about irreversible signs of disease evidenced in multiple physiological systems, just as high schizotypal characteristics bring about signs of psychotic illness across multiple physiological and psychological domains). Claridge[26] argued that both systemic and mental diseases could be seen to arise from a breakdown in the otherwise normal functioning of a biological system, rather than as an affliction imposed on the body. A second shared quality reflects the continuity between adaptive and maladaptive functioning of the system, given arbitrary cut-off

points for determining abnormality. Thirdly, both systemic and mental diseases may have multiple causes; in the case of hypertension, a number of environmental factors (like smoking, diet, and stress) may contribute to aberrant, sustained high blood pressure. Similarly, a variety of factors including genetic, psychosocial, and adverse life experiences may contribute to psychological illness vs health. In summary, Claridge[26] (p. 11) argued: "*the genetically influenced variations in brain organization which underlie temperamental and personality differences […] can be construed as dispositions to varying forms of mental disorder; and that the emergence of such disorder is, in essence, a transformation of these biological dispositions into signs of illness. […] It is only at the extremes that the disease 'entities' of psychiatry become clearly definable*."

Claridge's *fully* dimensional model of schizotypy, thus, spans a multidimensional set of personality traits (which can be loosely mapped to the symptoms of positive, negative and cognitive/disorganized features of psychotic illness) and is relevant to the spectrum of clinical conditions associated with psychotic features (eg, schizophrenia, schizoaffective disorders, affective psychotic disorders) in championing the view of *Einheitspsychose*.[27] As such, the traits underlying the schizotypy construct are proposed to vary along continuous dimensions in the general population and are not necessarily linked to psychopathology; transition to illness is influenced by a wide range of biological and psychological factors (not restricted to genetic influences), and the range of psychopathology encompasses functional psychoses and disorders of personality. Claridge's *fully* dimensional model can, therefore, be viewed upon as an extension of the Eysenckian model, considering schizotypy or psychosis-proneness to be continuously distributed in the population and a necessary but not sufficient condition for the development of psychotic illness. As such, it incorporates aspects of the *quasi*-dimensional model within the high-schizotypy spectrum, but suggests the issue of clinical relevance to be a factor of a second dimension (health), rather than inherent of schizotypy/Psychoticism (as were the views of Meehl and Eysenck). Thus, within Claridge's model there lies the potential for the existence of "happy schizotypes"[28] or "benign schizotypy"[29], ie, persons who score extremely high on measures of positive schizotypy, but are below the population-average in negative and cognitive/disorganized schizotypy[30] and, therefore, experience their psychotic-like experiences as rewarding and enhancing regarding their life satisfaction. Conversely, healthy offspring of schizophrenic patients have been shown to have above-average values in negative and cognitive/disorganized schizotypy, but below-average positive schizotypal traits.[31] It appears, therefore, that "benign/happy schizotypy"[28,29] only refers to a combination of high positive and simultaneously low negative and cognitive/disorganized traits, while the co-occurrence of high values in all schizotypy facets is highly predictive of schizophrenia.[32,33] This notion is substantiated by the finding that genetic risk scores for schizophrenia are inversely related to psychotic-like experiences and psychometric measures of positive schizotypy in healthy individuals[34,35] and the converse finding[36] (and unpublished data from Schultze-Lutter) that while negative but not positive schizotypy is highly predictive of clinical high risk for schizophrenia, it is the newly accrual of positive symptoms that ultimately leads individuals from clinical high-risk populations to seek professional help. In other words, "benign schizotypy" and clinical high risk may constitute opposite sides of the same coin, namely, high values in one but not another schizotypy facet.

Additionally, we find it helpful to point out that Claridge's model also relies heavily on a different understanding of the term "psychosis." It is commonplace to consider "psychosis" as inherently of clinical relevance, whereby Claridge is often criticized for his view, that there may exist a state of "healthy psychosis." It is noteworthy, however, that both historically (eg, Aristotle and Plato[37]) and etymologically (q.v., OED.com) the concepts of madness and psychosis are not necessarily linked to illness. Claridge's understanding of the term psychosis is, therefore, surely uncommon within clinical sciences, but also not untenable (figure 1).

The Importance of Conceptual Clarity

With increasing interest in neurodevelopmental models of psychotic disorders, it is important that researchers heed the distinctions between these models in order to clarify the meaning of terms like schizotypy or psychosis-proneness—even psychosis itself—when using them to denote risk for disorder, or otherwise. That is, it should be clearly articulated which *framework* the research is being conducted within since the concepts of "schizotypy" or "psychosis-proneness" are not identical among these model. For example, in studies of the general population where subgroups are operationally defined by their range of scores on measures of "schizotypy," it may be uncritically accepted that a "schizotypy" group is synonymous with what Meehl defined as schizotypal (or they may be referred to as "psychosis-prone" when there is very low likelihood that they may ever transition to clinical psychosis; these are but some interpretations that could arise). At first glance, it may not be obvious as to the importance of clarifying these finer points of distinction, but with multiple measures now available to psychometrically assess schizotypy, the different theoretical backgrounds from which these scales arose are highly relevant to their interpretation in modern studies. However, this by no means implies that scales derived to measure "Meehl's schizotypy" cannot be used to measure "Claridge's schizotypy", or vice versa; it is for this precise reason that researchers should be aware of the theoretical distinctions behind their construction, and what it

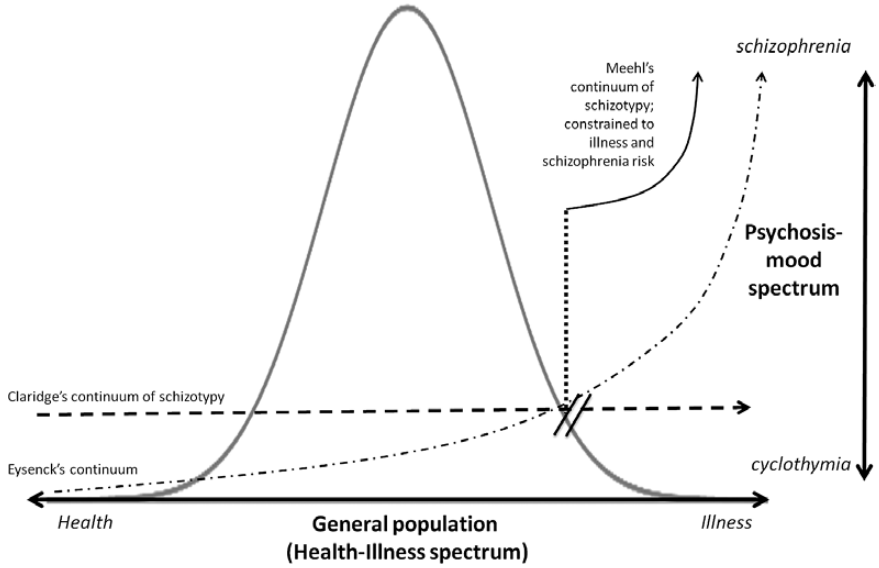

Fig. 1. Schematic of Meehl's, Eysenck's, and Claridge's continuum models of risk for psychosis spectrum disorders, mapped on 2 axes representing separate dimensions of *illness-health* and the *psychosis-mood spectrum.* Within *Meehl's schizotypy model* (solid line), the discrete taxon of schizotypy exists as 10% of the general population and is underpinned by an inherited, integrative neural defect (schizotaxia). Within *Eysenck's model* (dotted-and-dashed line), risk for schizophrenia is seen as a monotonic function of the personality dimension of Psychoticism; extreme values in Psychoticism represent psychotic disorder, and individual variation in an independent dimension of cyclothymia/schizothymia is said to explain differences within the group of psychotic disorders. Within *Claridge's fully dimensional model* (dashed line), schizotypy is seen as a set of behaviors and characteristics distributed normally in the general population, with the potential for illness arbitrarily distinguished at the extreme end of the health-illness spectrum. Like Eysenck, Claridge proposes that variance within the psychotic disorders (ie, within the psychosis spectrum) would be explained by other dimensions of personality (not shown here).

may mean for certain members of the general population to score highly on them, their scope (in terms of subdomains assessed) and potential to yield certain results in, eg, factor or latent class analyses.

For example, the content and style of psychometric measures of schizotypy have varied according to the investigators' aims and theoretical standing. The earliest scales (Wisconsin Schizotypy Scales; WSS) focused on measurement of vulnerability for *specific symptoms* of schizophrenia, including perceptual aberration,[38] magical ideation,[39] as well as physical and social anhedonia.[40] Other psychometric scales tap into hypomanic personality traits,[41] predisposition to hallucination,[42] delusions,[43] paranoia,[44] and schizotypal cognitions.[45] Yet other scales have been formulated on the basis DSM conceptualizations of "schizotypal personality" (the Schizotypal Personality Questionnaire, SPQ)[46] and/or "borderline personality" disorders,[47] or by assuming the existence of fundamental components like the asocial element of "Psychoticism."[25] In contrast, recent development of psychometric scales tapping the general schizotypy construct has been based on the empirically observed factor structure of schizotypal traits.[27,48–50] The origin of these scales bears relevance to their utility for particular research questions. While all pertinent measures are designed to capture "schizotypy," each was developed under the assumption of a different model and with different aims, such that their results should be interpreted accordingly: The WSS were modeled in light of the Meehlian model and include items "*transparently concerned with psychopathology*"[51] (p. 181), while the authors of the more recently developed O-LIFE generally attempted to avoid items of extremely high or low difficulty.[49] Thus, while these measures reflect different conceptualizations regarding the dimensionality of schizotypy, the relative likelihood of endorsing particular items on these instruments may affect the interpretation of scores in clinical or general populations and is likely to influence the results of taxometric analyses. The SPQ[46] was originally developed as a self-report screening tool for schizotypal personality disorder (which is undoubtedly not identical to schizotypy[15]). The factor structure of both WSS and SPQ was, therefore, originally not aimed at capturing truly disorganized aspects of schizotypy: The WSS were developed at a time when Meehl placed greater emphasis on anhedonia rather than cognitive slippage as the core feature of schizotypy,[4,11] and the SPQ scales "odd behavior" and "odd speech" are conceptually more related to eccentricity than cognitive disorganization. The O-LIFE,[27] on the other hand, was developed in accordance with Claridge's model and includes a disorganization scale (CogDis) and an impulsive nonconformity scale.

It becomes apparent that not only do the different conceptualizations of schizotypy differ regarding their core assumptions of the nature of the link between personality

and schizophrenia, but that the finer points regarding what should be understood as "core" schizotypy dimensions may vary according to the theoretical model from which a scale has been constructed. Additionally, comparing the commonly used schizotypy inventories (WSS, SPQ, and O-LIFE) shows that—while all of these encompass a positive, negative, and disorganized dimension—they differ slightly regarding their specific content: The "disorganized" dimension of the SPQ,[46,52,53] eg, is more closely related to "eccentricity" (scales: odd behavior and odd speech), while the respective scale "Cognitive Disorganisation" of the O-LIFE is more related to cognitive slippage or formal thought disorder. Pertaining to schizotypal traits, the adjective "cognitive," on the other hand, is also found in the positive (aka cognitive/perceptual) facets of the SPQ and the WSS, but here the adjective "cognitive" more closely resembles delusional thinking (rather than formal though disorder as in the O-LIFE).

Researchers should therefore be clear about whether their measurement of schizotypy is to be understood as an index liability for schizophrenia *only*, liability for all psychotic disorders, or liability for "*psychosis in schizophrenia*"[54] *and/or psychosis in* other non-neurological disorders or even the otherwise healthy (eg, as a function of psychotomimetic substances[55,56]). Moreover, researchers should be clear on whether they are testing a model in which there are circumstances given which proneness for psychosis in the general population *may* become pathological (ie, consistent with Claridge) or whether all forms of schizotypy are regarded as *abnormal* personality traits (ie, consistent with Meehl). This potential distinction between the existence of "normal" and "abnormal" personality features has yet to be fully resolved.

Summary and Conclusions

Although there is widespread consensus that a personality framework exists that is related to psychotic disorders and psychotic/psychotic-like experiences in other illnesses or even the otherwise healthy, a number of aspects of the liability models remain to be agreed upon. A great amount of disagreement can be traced back to subtle but crucial differences in conceptualization of health and disease, with implications for the concept of "schizotypy" as liability for schizophrenia or rather as proneness to unusual experiences and beliefs that are commonly experienced in the general population. Despite these major point of disagreement, there is arguably some consensus insofar as risk for schizophrenia is likely caused by a complex interaction of genetic and environmental influences and is primarily represented through cognitive disorganization and negative facets of schizotypy (rather than positive schizotypy). This notion is consistent with recent findings that polygenic risk scores for schizophrenia are inversely associated with positive dimensions of schizotypy *in healthy individuals*.[34,35]

The most prominent issue to be resolved concerns whether the multidimensional construct of schizotypy should be regarded as expressions of normal variation in functioning (ie, normally distributed among the general population) in a manner that precludes the distinction of a discrete taxon of individuals at highest risk for schizophrenia (or other psychotic disorders) or whether these concepts (continua and taxon) are actually compatible such that *both* may be true of the construct of schizotypy. The latter notion suggests that, rather than a true taxon, qualitative entities (eg, schizophrenia, but also "clinical high risk" or "benign schizotypy") may be focal points or observed constellations of several traits (ie, taxon-like clusters). This would be in line with original interpretations of "types" and "syndromes" by Kretschmer and Eysenck[19] and has also been suggested by other authors (eg, Gale et al,[57] Grant,[9,58] Mason[59]). Similarly, a comprehensive review of the dimensionality of schizophrenia symptoms[60] concludes that although (at first glance) the majority of taxometric research calls into question the dimensional distribution of schizophrenia symptoms in the general population, serious methodological flaws often challenge the validity of these findings, and that the dimensionality of *schizotypy* remains to be adequately tested. When introducing variables commonly associated with schizophrenia additionally to schizotypy data (eg, schizophrenia-related genetic polymorphisms, cannabis use, obstetric complications, familial risk); however, a clear taxonic pattern emerges.[61] We, thus, suggest that—while relevant facets of personality (gathered under the wide rubric of "schizotypy") may be individually dimensional in nature—risk-for-schizophrenia is not, but rather likely to be represented in the co-occurrence of several highly "schizotypal" traits, forming a taxon-like cluster.

It is not the major aim of this review, however, to argue for an inherently correct, single solution. Primarily, we aim to illustrate the importance of conceptual clarity and to encourage researchers not only to keep in mind the model that they are working within but also to—perhaps most importantly—place their research findings within the scope of the contending models and discuss the implications regarding the models' verisimilitude. We believe that only with such increased clarity and acknowledgment of these issues will there be substantial progress in determining the status of "schizotypy" on the path to clinical psychotic states and related psychopathology.

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